

Transvenous Embolization of a Ruptured Deep Cerebral Arteriovenous Malformation

A Technical Note

V.M. PEREIRA¹, A. MARCOS-GONZALEZ¹, I. RADOVANOVIC², P. BIJLENGA²,
A.P. NARATA¹, J. MORET³, K. SCHALLER², K.O. LOVBLAD¹

¹ Interventional Neuroradiology Unit, Neuroradiology Service, ² Neurosurgery Service, University Hospital of Geneva; Geneva, Switzerland

³ Interventional Neuroradiology Service, Beaujon CHU; Paris, France

Key words: arteriovenous malformation, DMSO, Onyx, squid, transvenous

Summary

Ruptured cerebral arteriovenous malformations (AVMs) usually require treatment to avoid re-bleeding. Depending on the angioarchitecture and center strategy, the treatment can be surgical, endovascular, radiosurgical or combined methods. The classic endovascular approach is transarterial, but sometimes it is not always applicable. The transvenous approach has been described as an alternative for the endovascular treatment of small AVMs when arterial access or another therapeutic method is not possible. This approach can be considered when the nidus is small and if there is a single draining vein. We present a technical note on a transvenous approach for the treatment of a ruptured AVM in a young patient.

Introduction

Cerebral arteriovenous malformations (AVMs) present a cumulative hemorrhagic risk of 2% annually, and sometimes have an even higher incidence, depending on the AVM angioarchitecture and patient history¹. The management of AVMs is still controversial, but a previous hemorrhage is associated with an increased rupture risk². Different treatment options have been proposed, including surgery, endovascular treatment, radiosurgery or a combined treatment³.

Endovascular treatment usually has two strategies: staged and targeted treatment using

glue⁴ or Onyx (aiming to decrease the hemorrhagic risk or decrease the AVM size for secondary treatment with surgery or radiosurgery) or curative treatment with complete exclusion of the AVM nidus. The classic and regular endovascular approach is transarterial access with the selective catheterization of an arterial feeder as close as possible to the nidus and subsequent injection of the embolic agent. Nevertheless, there are some cases in which arterial access is not possible and other treatment methods are not applicable or carry a high risk for the patient⁵. The transvenous approach has been described as an alternative for the endovascular treatment of small AVMs when arterial access is not possible. This approach can be considered when the nidus is small and if there is a single draining vein⁶. We present a technical note on a transvenous approach for the treatment of a ruptured AVM in a young patient recently treated during the LINNC course - Paris 2012.

Case Report

History

A 31-year-old patient without a medical history presented at the emergency department with explosive headaches, followed by nausea and vomiting.

Upon admission, his CT scan showed a prominent right temporo-occipital hematoma, with intraventricular hemorrhage and hydro-



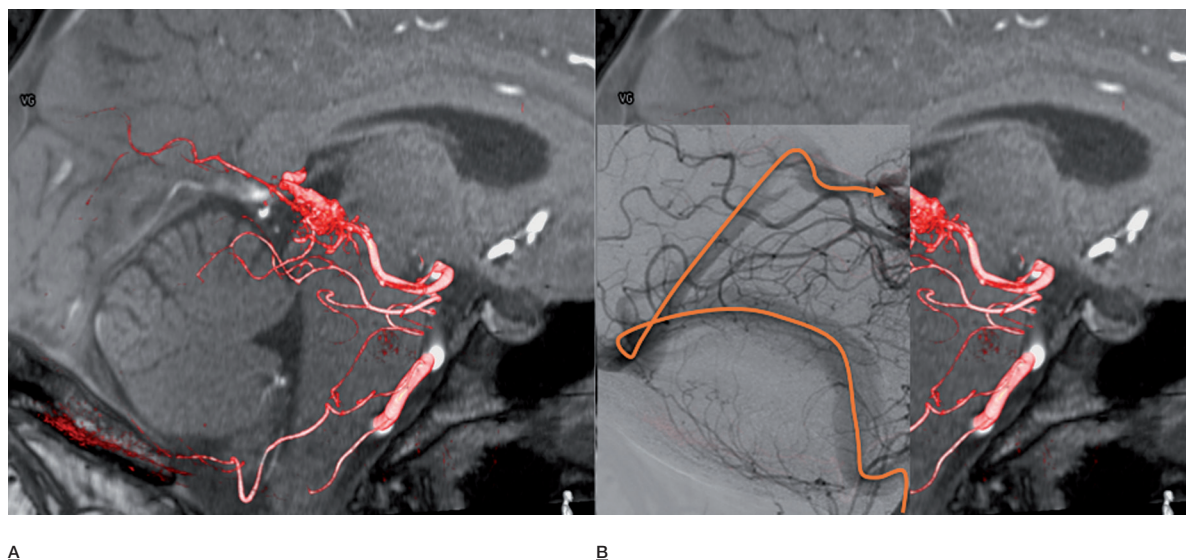
Figure 1 Initial imaging assessment. A) CT non-contrast scan showing the initial aspect of the hemorrhage with intraventricular extravasation. B-D) Digitally subtracted angiogram with 3D (B), AP (C) and lateral (D) views showing the AVM presenting a 2.5 cm nidus fed by multiple small posterior choroid branches from the right posterior cerebral artery draining to a ventricular vein that joins the internal cerebral vein just before the Galen vein.

cephalus. CT angiography showed an image compatible with an AVM located at the medial wall of the occipital horn of the right lateral ventricle (Figure 1). Ophthalmologic examination showed that the visual field was normal.

A cerebral angiogram confirmed the diagnosis, showing a small AVM with a 2 cm nidus whose feeders essentially came from multiple posterior choroid branches of the right posterior cerebral artery (PCA). A single venous drainage was performed through a ventricular vein joining and then through the right internal

cerebral vein and the Galen vein toward the straight sinus (Figures 1 and 2).

After an interdisciplinary discussion with the neurosurgery team, a decision of conservative treatment during the acute period was made. Surgical treatment was not considered due to the deep location of the lesion. Radiosurgery was considered as presenting a moderate risk, but it was not the first choice because of the rupture presentation, the nidus circumscribing the PCA and proximity with the visual pathways.

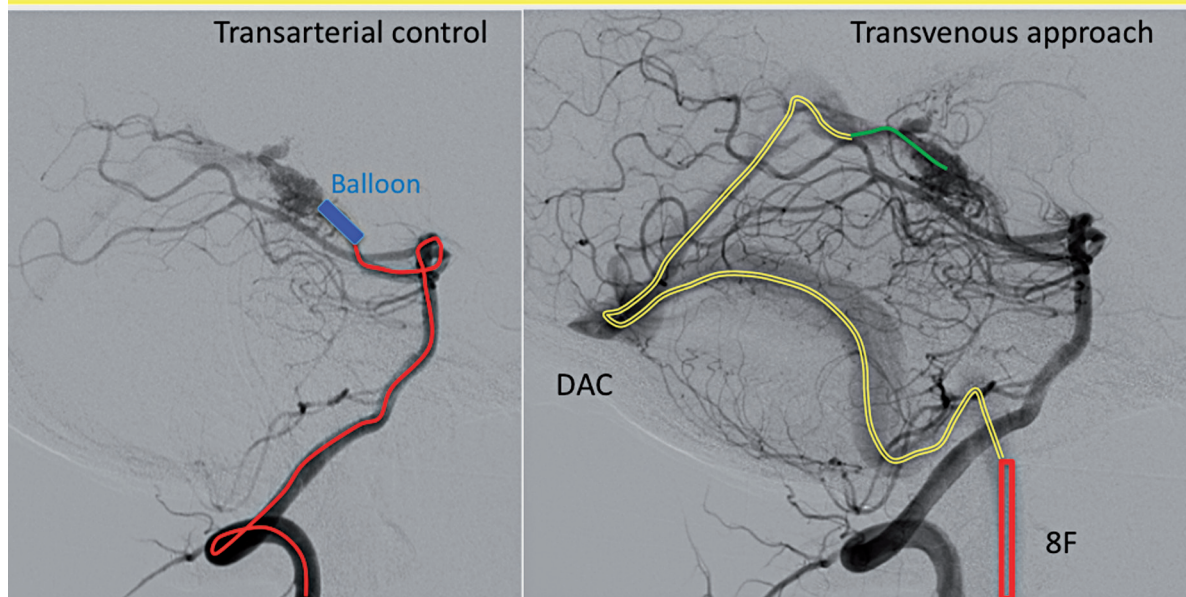


A

B

Figure 2 Strategic planning of the procedure. A) MRI and 3DRA fusion showing the location of the AVM nidus and the ventricular and arterial system around the lesion. B) Superposition of the venous phase DSA – lateral vein on the MRI/3DRA fusion showing the venous route for the AVM approach.

Treatment Strategy



A

B

Figure 3 DSA with early arterial (A) and venous phase (B) lateral views. Schematic planning of the procedure. A) Positioning of the system at the arterial side with the balloon at the PCA, permitting arterial lumen protection and assisting in the transvenous injection of the DMSO polymer by decreasing the AVM flow during the procedure. B) Transvenous approach with the 8F guiding catheter located at the level of the jugular vein, the distal access catheter (DAC) 4F located at the end of the straight sinus and the microcatheter on the venous system as close as possible to the nidus.

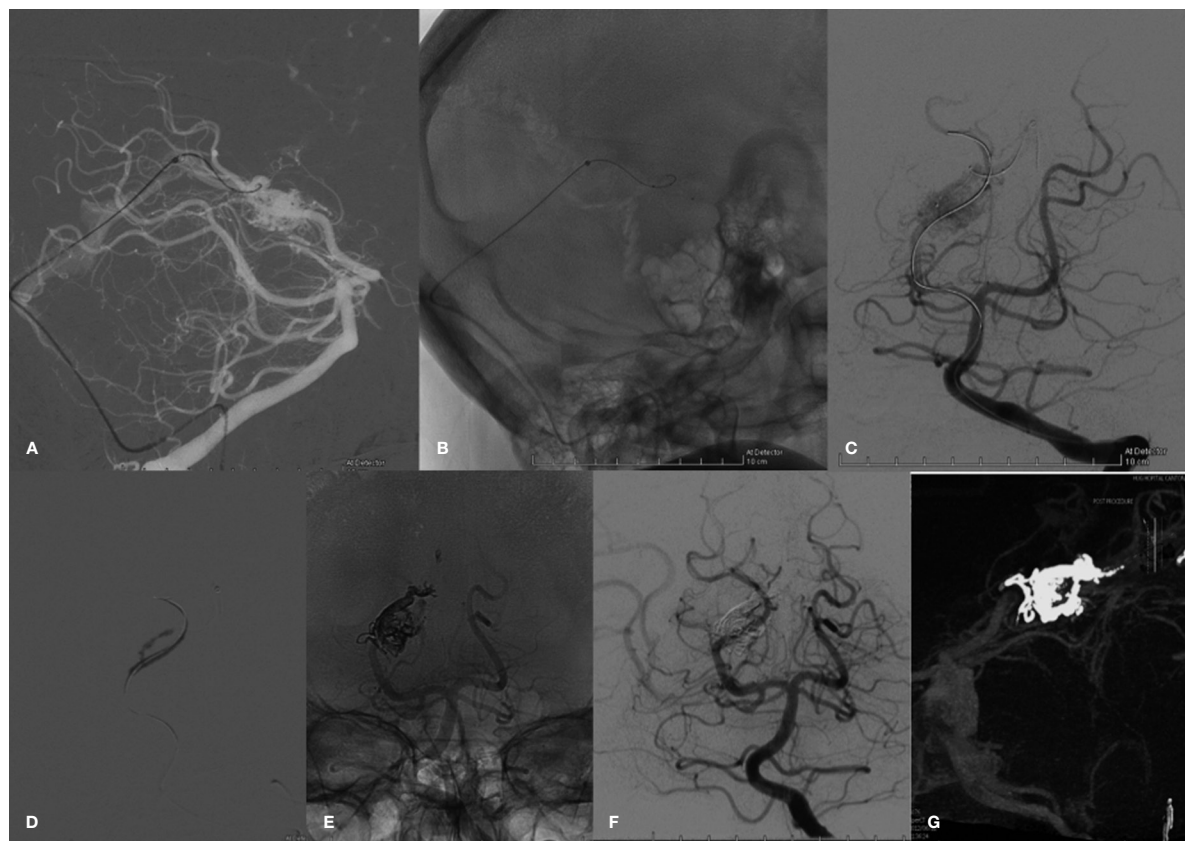


Figure 4 Procedural images from DSA, A,B) Lateral view, late arteriovenous phase roadmap demonstrating the radio-opaque microcatheter route through the venous system to the ventricular vein close to the AVM nidus. C-F) DSA working projection, oblique view. C) Initial DSA, arterial phase showing the AVM before embolization. D) Subtracted image of the initial onyx injection and the plug formation on the venous side. Note also the balloon inflation at the PCA covering the arterial feeders through the AVM. E, F) Final DSA control angiogram, subtracted and non-subtracted views demonstrating the DMSO polymer cast corresponding to the initial AVM nidus and complete AVM occlusion and the patency of the PCA artery. G) VasoCT (Xpert CT tool for post-interventional control imaging) showing the polymer cast on the AVM nidus around the artery.

Technical Note

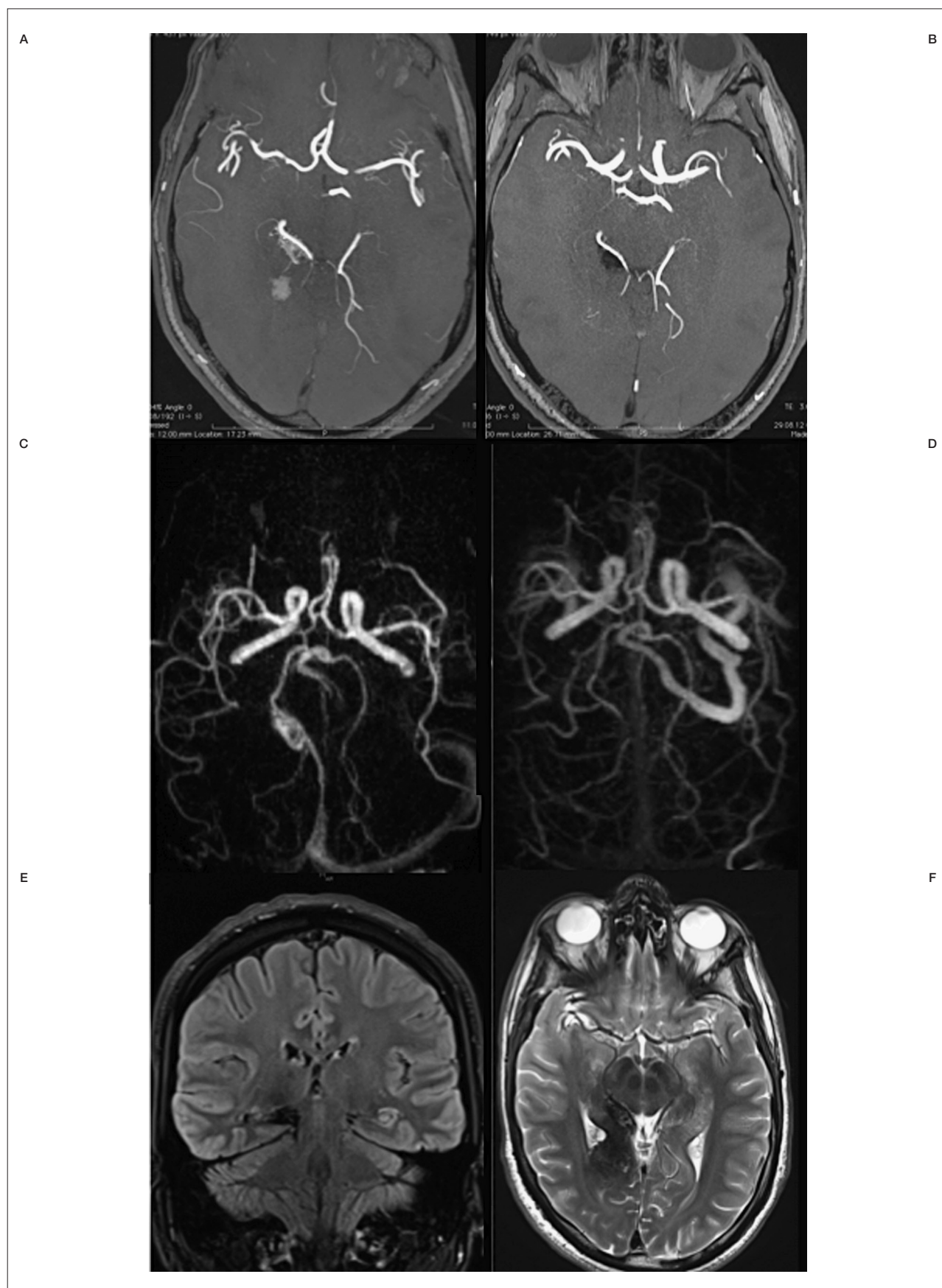
Venous access

An 8F femoral sheath was used at the right femoral vein. An 8F-guiding catheter (Softip, Boston Scientific, Plymouth, USA) was placed in the right jugular vein. A distal access catheter (DAC 44/130 cm, Concentric Medical, Mountain View, CA, USA) was placed in the distal part of the straight sinus just before the Galen vein. A dimethyl sulfoxide (DMSO)-compatible 1.2F (Sonic, BALT, Montmorency, France) microcatheter with a detachable tip of 1.5 cm was navigated with the aid of a 0.007 microwire (Hybrid, BALT, Montmorency, France) to the ventricular draining vein close to the AVM nidus (Figure 2).

Arterial control

A 6F long sheet (Destination, Terumo, Elkton, USA) was placed into the left subclavian artery, and then coaxially an intermediate 6F catheter (Reflex, Covidien, Irvine, USA) was placed in the left vertebral artery. The guiding catheter was flushed with saline via a pressure bag. A Hyperglide 4×20 mm balloon (Covidien, Irvine, USA) was placed into the right PCA, covering the multiple “en passage” small posterior choroidal feeders.

Figure 5 Pre- (A,C) and 2 months post-operative (B,D) MRI scans. A,B) Axial images, TOF MRA: complete occlusion of the AVM nidus and permeability of the artery. C,D) Axial views of dynamic contrast MRA (TWIST): complete occlusion of the AVM. E) Coronal FLAIR images; F) axial T2 images: complete resolution of the perilesional edema after the hemorrhage.



The strategy

The aim of placing the balloon on the arterial side was to facilitate the retrograde reflux of the embolic agent (Squid 18, Emboflu, Fribourg, Switzerland) into the nidus and protect the patency of the PCA. SQUID is an ethylene vinyl alcohol (EVA) copolymer dissolved in DMSO polymer at different concentrations: 4.9% and 6.0%. Micronized tantalum powder (35% wt/vol) was added for radiopacity. The vials were kept on a shaker (Vortex-Genie, Scientific Industries, Bohemia, NY, USA) for at least 20 minutes to ensure stable mixing of the tantalum with the polymer (Figure 3).

We began the procedure by preparing the microcatheter with an injection of 0.25 mL DMSO to fill the dead space, and the embolic agent was then injected progressively. At the same time as the polymer injection, we inflated a temporary balloon at the right PCA. Temporary occlusion of the arterial supply permitted the creation of a “plug” in the draining vein, avoiding a long reflux into the internal cerebral vein or Galen vein (Figure 4). During injections, we were able to pause, obtain an angiogram to assess nidus occlusion and the status of the draining vein and PCA artery, and then continue with the polymer injection.

We injected 3 cc of the embolic agent (SQUID, Emboflu, Fribourg, Switzerland) over 16 minutes, with complete occlusion of the nidus retrograde. The procedure was completed, and a VasoCT (Philips) showed the embolic material completely inside the nidus and circumscribing the right PCA. No reflux into the deep venous system was observed. To remove the microcatheter, we advanced the DAC to the Galen vein and began the withdrawal of the microcatheter to the detachment of the proximal part and the distal tip. There was no retraction of the venous system with this technique.

The patient received 5000 UI of heparin during the procedure and was extubated just after the procedure. No hemorrhagic or embolic complications were observed during or after the intervention. He underwent intensive control of blood pressure (BP) for 24 hours with invasive monitoring control and anti-hypertensive medication to keep the mean BP below 80 mmHg. The patient's evolution during the early post-operative course was unremarkable. The patient underwent a follow-up MRA two months after the procedure, which

showed complete occlusion of the AVM (Figure 5), patency of the right PCA and the absence of edema or other abnormal signals, except for the small sequelae of a hematoma at the collateral trigone of the lateral ventricle. The patient had an mRS score of 0, and the ophthalmologic control examination was also normal.

Discussion

While the treatment of unruptured AVMs is still controversial, there is a consensus that a previously ruptured AVM presents an increased hemorrhagic risk and should be considered for treatment². Endovascular treatment is one of the therapeutic options available for AVM treatment. Among the different endovascular strategies, partial targeted or complete AVM occlusions, the choice depends mostly on the center's experience and the embolic material. Endovascular treatment using NBCA presents complete occlusion rates in the range of 5 to 15% in general, but some exceptional series have reported up to 40%¹⁴. The use of EVA/DMSO polymers (Onyx or SQUID) has shown occlusion rates from 20 to 27.7% for sometimes high Spetzler-Martin grade AVMs.

The regular endovascular approach to an AVM is transarterial embolization. Depending on the arterial feeders, this route permits a selective approach, allowing good penetration into the nidus and good control of the venous penetration if the nidus is not completely occluded. The strategy depends on the AVM size, location, and angioarchitecture. When an endovascular arterial approach is not possible, other therapeutic modalities (surgery and radiosurgery) should be considered. Recently, an endovascular transvenous approach for pial AVMs was described, and it could be an option in cases in which no other modality could be offered for clearly indicated AVM treatment.

The strategy behind this approach follows the usual thinking while using EVA/DMSO polymers from a transarterial approach: the final procedure for a complete occlusion of the AVM is performed with the occlusion of the draining vein, permitting a final retrograde reflux into the nidus to assure complete occlusion of the AVM. The strategy behind the transvenous approach is the same, except that the access is from the venous side directly. Indeed, this approach cannot be offered in every case.

Ideal cases are AVMs with a small nidus (< 3 cm), the absence of safe arterial access and only one draining vein present.

The venous approach for AVMs was previously proposed for AVM treatment for venous sclerotherapy under induced hypotension, but there was no follow-up after the initial experience²⁰. The transvenous approach has been used in some French centers with Onyx, but the first study was performed by Nguyen et al.¹⁰ and followed by Kessler et al. with a larger series of patients⁹.

The present case report adds different technical aspects that increase the safety of this approach compared to the previous reports. The presence of a balloon on the arterial side protects the arterial lumen in the case of “en passage” branches and permits a flow decrease on the draining vein, avoiding migration and lamination of the polymer to the deep venous system. It also helps the formation of a “plug” around the tip of the microcatheter and retrograde reflux into the nidus. We also introduced the use of detachable tip microcatheters, which considerably increase the injection time and safety during withdrawal of the microcatheter. Furthermore, the use of the distal access catheter facilitated the recovery of the microcatheter, decreasing the retraction on the deep venous system. This is an important measure

when we consider navigation and further retraction on the deep venous system and the risk of hemorrhage due to venous injury.

This approach requires significant experience in the treatment of AVMs using EVA/DMSO polymers. Furthermore, advanced anatomical knowledge and careful analysis of the venous and nidal anatomy is required to determine security limits for polymer reflux. This is the third case of an AVM treated using the transvenous approach of the first author (VMP). The other cases performed in our hands before this case were uneventful and we describe this case in particular because of the technical set-up used that improved the safety of the procedure.

We emphasize that this approach should be used only as an alternative in cases in which no other therapeutic modality can be offered to some AVMs clearly indicated for treatment. A small nidus and a single draining vein are major architectural characteristics in favor of this approach. Criteria revision of the anatomy, 3D angiogram and DSA and MRI fusions can help in strategic and technical planning. When well indicated, this approach can be an alternative treatment of some cases and should be performed in centers with long-standing experience in the endovascular management of AVMs.

References

- 1 da Costa L, Wallace MC, Ter Brugge KG, et al. The natural history and predictive features of hemorrhage from brain arteriovenous malformations. *Stroke*. 2009; 40: 100-105.
- 2 Stapf C, Mast H, Sciacca RR, et al. Predictors of hemorrhage in patients with untreated brain arteriovenous malformation. *Neurology*. 2006; 66: 1350-1355.
- 3 Wedderburn CJ, van Beijnum J, Bhattacharya JJ, et al. Outcome after interventional or conservative management of unruptured brain arteriovenous malformations: a prospective, population-based cohort study. *Lancet Neurol*. 2008; 7: 223-230.
- 4 Meisel HJ, Mansmann U, Alvarez H, et al. Effect of partial targeted N-butyl-cyano-acrylate embolization in brain AVM. *Acta Neurochir*. 2002; 144: 879-887; discussion 888.
- 5 Saatci I, Geyik S, Yavuz K, et al. Endovascular treatment of brain arteriovenous malformations with prolonged intranidal Onyx injection technique: long-term results in 350 consecutive patients with completed endovascular treatment course. *J Neurosurg*. 2011; 115: 78-88.
- 6 Mounayer C, Hammami N, Piotin M, et al. Nidal embolization of brain arteriovenous malformations using Onyx in 94 patients. *Am J Neuroradiol*. 2007; 28: 518-523.
- 7 Spelle L, Mounayer C, Piotin M, et al. Intracranial arteriovenous malformations: review of epidemiologic and genetic data. [Article in French]. *J Neuroradiol*. 2004; 31: 362-364.
- 8 Thines L, Dehdashti AR, da Costa L, et al. Challenges in the management of ruptured and unruptured brainstem arteriovenous malformations: outcome after conservative, single-modality, or multimodality treatments. *Neurosurgery*. 2012; 70: 155-161; discussion 161.
- 9 Kessler I, Riva R, Ruggiero M, et al. Successful transvenous embolization of brain arteriovenous malformations using Onyx in five consecutive patients. *Neurosurgery*. 2011; 69: 184-193; discussion 193.
- 10 Nguyen TN, Chin LS, Souza R, et al. Transvenous embolization of a ruptured cerebral arteriovenous malformation with en-passage arterial supply: initial case report. *J Neurointerv Surg*. 2010; 2: 150-152.
- 11 Matsubara S, Mandzia JL, ter Brugge K, et al. Angiographic and clinical characteristics of patients with cerebral arteriovenous malformations associated with hereditary hemorrhagic telangiectasia. *Am J Neuroradiol*. 2000; 21: 1016-1020.
- 12 Lundqvist C, Wikholm G, Svendsen P. Embolization of cerebral arteriovenous malformations: Part II-Aspects of complications and late outcome. *Neurosurgery*. 1996; 39: 460-467; discussion 467-469.
- 13 Viñuela F, Duckwiler G, Jahan R, et al. Therapeutic management of cerebral arteriovenous malformations. Present role of interventional neuroradiology. *Interv Neuroradiol*. 2005; 11: 13-29.
- 14 Valavanis A, Yasargil MG. The endovascular treatment of brain arteriovenous malformations. *Adv Tech Stand Neurosurg*. 1998; 24: 131-214.
- 15 Weber MA, Ahlhelm F, Essig M, et al. Treatment options for arteriovenous malformations. [Article in German]. *Radiologe*. 2007; 47: 893-899.
- 16 Panagiotopoulos V, Gizewski E, Asgari S, et al. Embolization of intracranial arteriovenous malformations with ethylene-vinyl alcohol copolymer (Onyx). *Am J Neuroradiol*. 2009; 30: 99-106.
- 17 Abud DG, Riva R, Nakiri GS, et al. Treatment of brain arteriovenous malformations by double arterial catheterization with simultaneous injection of Onyx: retrospective series of 17 patients. *Am J Neuroradiol*. 2011; 32: 152-158.
- 18 Wikholm G, Lundqvist C, Svendsen P. Embolization of cerebral arteriovenous malformations: Part I-Technique, morphology, and complications. *Neurosurgery*. 1996; 39: 448-457; discussion 457-449.
- 19 Weber MA. Arteriovenous malformations of the brain: What is worst - the disease or the cure?. [Article in German]. *Radiologe*. 2007; 47: 661.
- 20 Massoud TF, Hademenos GJ. Transvenous retrograde nidus sclerotherapy under controlled hypotension (TRENSh): a newly proposed treatment for brain arteriovenous malformations-concepts and rationale. *Neurosurgery*. 1999; 45: 351-363; discussion 363-355.

Vitor Mendes Pereira, MD, MSc
 Head of Interventional Neuroradiology
 University Hospital of Geneva
 Rue Gabrielle-Perret-Gentil, 4
 1211 Geneva, Switzerland
 E-mail: vitor.mendespereira@hcuge.ch